

## Summary and Conclusion

Vitiligo is a depigmented disorder characterized by the loss of melanocytes from the cutaneous epidermis. Although the exact etiology of vitiligo has not yet been established, the abnormal immune responses frequently observed in vitiligo patients have led to the suggestion that, in some cases, the condition has an autoimmune component.

Circulating autoantibodies and autoreactive T cells that recognize pigment cell antigens have been detected in the sera of a significant proportion of vitiligo patients compared with healthy individuals. In addition, vitiligo is often associated with other disorders that have an autoimmune origin, including Hashimoto's thyroiditis, type 1 insulin-dependent diabetes mellitus and others.

→ Furthermore, effective use of immunosuppressive therapies to treat vitiligo; the association of vitiligo with certain major histocompatibility <sup>Antigens</sup> have all added credence to the hypothesis that immune reactions play a role in vitiligo pathogenesis.

Forty patients were enrolled in the study (30 females and 10 males). Complete history taking, dermatological and general examination aided in vitiligo diagnosis. Different surgical modalities combined with PUVA therapy were used in attempt to induce repigmentation in the vitiliginous areas and comparing these results with those of PUVA alone.

Blood samples were taken from each patient before and after different surgical modalities combined with PUVA therapy and then compared with control subjects, which were also included in the study (10 controls).

Treating localized vitiligo with epidermal grafting using cryo-induced blisters is an incorrect procedure, as we used the least cooling cryogen (Freon 12) with a temperature of  $-60^{\circ}\text{C}$  and still it caused melanocytes destruction due to melanocytes sensitivity to temperature. Melanocytes die at a relatively high temperature of  $-5^{\circ}\text{C}$  to  $-10^{\circ}\text{C}$ , while keratinocytes need to be frozen to  $-50^{\circ}\text{C}$  for optimum destruction.

Suction induced blisters for epidermal grafting as a method of treatment in vitiligo patients revealed to have a placebo effect besides it is a waste of time, painful to the patient and have a quite big percent of epidermal rejection.

PUVA therapy has a satisfactory response on vitiligo patients, but patients have to attend three times a week for 12 to 24 months of continuous therapy or at least attend 150 settings.

Intact suction blisters revealed to be an excellent mechanism in hastening PUVA results or in areas with delayed response.)

Melanocytes are still present in the depigmented epidermis of patients of vitiligo even after stable disease of 25 years duration.

Melanocytes are never completely absent in the depigmented epidermis and that these melanocytes can reconvert their functionality in vivo or in vitro under certain circumstances

On attempt of wound healing melanocytes migrate to the injured epidermis in order to repigment it. Repigmentation in the recipient sites after graft rejection (by suction blisters for epidermal grafting) also indicates melanocytes migration, which was estimated to occur during the period of 7 days.

Preparing vitiliginous patients with PUVA before treatment by suction blisters is very beneficial as it induces keratinocytes and other inflammatory cells in vitiligo lesions to release various melanocyte growth factors to stimulate the inactive melanocytes reservoirs.

PUVA therapy deplete vitiligo-associated melanocytes antigens, decrease the antigenic potential of antibody directed against melanocytes and decrease the density of Langerhans cells in treated skin; PUVA mediate repigmentation in part through an immunological process.

Soluble ICAM-1 levels pretreatment versus post treatment and sICAM-1 pretreatment versus s ICAM-1 in control were statistically significant,  $p < 0.005$ . These findings propose a considerable role of an autoimmune involvement in the pathogenesis of vitiligo.

According to these results, sICAM-1 levels could be a marker in the course of vitiliginous lesions.